120 Anti-*Pseudomonas aeruginosa* IgY antibodies promote bacterial clearance in a murine pneumonia model

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Objectives: The present study explored the possible protective effect of IgY on PA lung infection in vivo.
Methods: In vivo model of acute lung infection: Balb/c mice were anaesthetized with isoflurane and PAO1 vaccine strain ± specific (S-IgY) or control (C-IgY) was inoculated intranasally. Mice were sacrificed after 2, 6 and 24h and lungs removed aseptically, weighted and suspended in PBS. A blinded observer engaged a clinical scoring system (0–5) of the mice. Lungs were homogenized, serially diluted and cultured on Conradi-Drigalski medium for estimation of bacterial load.
Results: *Relative lung weight*: Lung weights in the S-IgY treated group were significantly reduced 24h post-infection compared to PBS controls ($p < 0.03$). No significant difference between C-IgY and PBS groups were observed.
Clinical symptom score: The clinical score was significantly lower in the S-IgY group compared to controls after 6h (C-IgY: $p < 0.05$, PBS: $p < 0.05$). After 24h the clinical score in the S-IgY was reduced additionally compared to controls (PBS: $p < 0.002$, C-IgY: $p < 0.04$). No significant difference between C-IgY and PBS groups were observed.
Quantitative bacteriology: The bacterial load of S-IgY treated mice was significantly reduced 2h post-infection compared to PBS group ($p < 0.02$) and C-IgY ($p < 0.03$) and further reduced 6h post-infection compared to both control groups (PBS: $p < 0.0001$, C-IgY: $p < 0.03$). After 24h the lung bacteriology in S-IgY treated mice was reduced by 2 logs compared to PBS ($p < 0.0001$) and C-IgY ($p < 0.0002$) groups.
Conclusion: The present results imply that anti-PA IgY antibodies protects against PA lung infection due to readily bacterial clearance in the airways.

121 Anti-*Pseudomonas aeruginosa* antibodies and microbiological outcome in not chronically infected patients

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Objectives: *P. aeruginosa* (Pa) lung infections cause a lung function decline and a systemic increase of serum antibodies against Pa antigens. Eradication treatment can clear Pa in the early phases of the infection. The purpose of this study was to evaluate, as marker of early stages of Pa infection, the anti-Pa immune response in not chronically infected patients.
Methods: Serum from 153 not chronically infected patients (median age 10 years, range 0–46) in a regular follow-up in the period 2011–2013 were analyzed using Enzyme-Linked Immuno Sorbent Assay (ELISA) for the presence of IgG antibodies against Pa sonicated cell extract (St-Ag) (serogroups 1–17). Patients were classified according to their microbiological status (Leeds definition).
Results: During the observation period 2 (1.3%) patients became chronically infected. Patients never infected or free from Pa were respectively 48 (31.4%) and 60 (39.2%) of the total. In both groups antibody titre was under the cut-off level (1.50±1.90 EU). In the group of 43 (28.1%) patients, intermittently infected by Pa, the titer of anti-Pa antibodies was significantly higher in subjects presenting a concomitant bacterial isolation than those who did not showed Pa isolation (3.03 EU ± SD 3.20, 1.72 EU ± SD 1.7, respectively) ($P < 0.05$).
Conclusion: The evaluation of specific anti-Pa antibodies may be a useful way to monitor early Pa infection in patients with intermittent infection.

122 Modulation of cystic fibrosis gut inflammation – a pivotal role of the lung gut axis?

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Objectives: Recent studies have suggested a potential role for the lung-gut axis in the pathogenesis of gut inflammation in CF. Modulating lung inflammation by targeting the cycloxygenase pathway and prostaglandin E2 (PGE_2) can slow inflammatory pulmonary responses in people with CF. The objective of this study is to investigate if pro-inflammatory changes in the lung affect gut inflammation.
Methods: Plasma levels of PGE_2 in CF patients ($n = 25$) was detected. CF bronchial epithelial cells (ΔF508 homozygote, CFBE41o–) and human bronchial epithelial cells (16HBEo–) were seeded at 2×10^5 cells/ml. Cells were stimulated with PGE_2 and cell supernatant was harvested. HT29 colon epithelial cells were seeded at 2×10^5 cells/ml and treated with harvested supernatant. Cell proliferation of HT29 cells was determined and changes in cytokine levels of IL-6, IL-8 and TNFα were detected by ELISA.
Results: Supernatant from CF lung cells augmented IL-6, IL-8 and TNFα production by HT29 cells. Relative to HT29 cells cultured in normal bronchial 16HBEo– epithelial cells, secretion of IL-6, IL-8 and TNFα by HT29 cells incubated with CFBE41o– CF cell supernatant was significantly increased 2.7 fold, 1.8 fold and 1.4 fold respectively. Culture of the cells in PGE_2-treated CF cell supernatant relative to PGE_2-treated normal bronchial epithelial cells further increased IL-6, IL-8 and TNFα. There was no significant change in cell proliferation of HT29 intestinal epithelium cells.
Conclusion: This study shows that inflammatory changes in the CF lung can modulate cytokine production in the gut. Thus, independent of active CF lung infection this mechanism may regulate intestinal inflammation.

123 How frequent is *Clostridium* in our CF patients?

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Objectives: Children with cystic fibrosis are, unfortunately, candidates at multiple antibiotic courses, having a potential increase risk for pseudomembranous colitis with *Clostridium difficile*. The aim of the paper was to evaluate the frequency of *C. difficile* infection among children with cystic fibrosis (CF).
Methods: Retrospective analysis over a ten years period was done, using the information from our CF center's database. In all the patients, only presentation with diarrhea occurred during antibiotherapy were taken into consideration. Diagnosis test for *C. difficile* infection was performed by enzyme immunoassay for detection of toxins A and B.
Results: Over a ten years period, 308 patients with cystic fibrosis were admitted in our clinic; only five of them (1.62%) were diagnosed with *C. difficile* infection. Patients were diagnosed in the last 4 years, by the detection e of toxin A or toxin A and B (in 2 patients) in the presence of diarrhea; they had a favorable outcome, with a good response to treatment (metronidazole in 3 cases, metronidazole and vancomycin in 2 cases). All patients had chronic *Pseudomonas aeruginosa* infection and received more than fourteen days of antibiotics.
Conclusion: *Clostridium difficile* infection should be consider for evaluation in cystic fibrosis patients with diarrhea who receive antibiotics. Special attention is necessary when antibiotherapy is given for a long time, as commonly reccomended in cystic fibrosis patients.